HEART RATE: A PREDICTOR OF EARLY MORTALITY IN PATIENTS WITH MYOCARDIAL INFARCTION

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ABSTRACT

A number of epidemiologic studies have reported a positive relationship between heart rate, cardiovascular disease and mortality. To examine the correlation between heart rate and mortality after acute myocardial infarction (AMI), 2147 patients hospitalized in coronary care units in Isfahan were investigated in a cross-sectional study. Their heart rate was measured according to an electrocardiograph obtained during the 2nd, 5th and final days of hospitalization, and all patients were followed for cardiovascular mortality. The means of 3 measurements were divided into four fractions.

A strong positive relationship between heart rate and cardiovascular mortality was obtained. Confounding variables such as age, sex, type of infarction and drugs were adjusted by regression models. The nonlinear regression relationship was due primarily to a sharp increase in mortality in the highest quartile of heart rate.

We conclude that although sinus tachycardia can be considered as a predictive independent risk factor for mortality after AMI, further longitudinal studies are required.

Keywords: heart rate, myocardial infarction, mortality, prognosis, sinus tachycardia, cardiovascular diseases, risk factors.

INTRODUCTION

It is believed that a low heart rate is associated with longevity, largely because of lower rates of cardiovascular mortality in patients with low heart rates. Increased heart rate is associated with increased risk of cardiovascular mortality.1,4

There is also evidence of a positive relation between resting heart rate and non-cardiovascular mortality,2,5,8 with a significant association between heart rate and cancer mortality.3,9 New data from the Framingham study showed that this association is seen in normotensive and hypertensive patients, and also indicate an association between heart rate and sudden cardiac death.4,10

Sinus tachycardia following AMI (acute myocardial infarction) is common and is frequently an unfavorable prognostic sign. Patients with a large area of infarcted myocardium may have sinus tachycardia due to left ventricular dysfunction; other causes are anxiety, fever, pain, pulmonary embolism, hypovolemia, anemia or drugs such as dobutamine, dopamine, etc.16 Commonly, the initial episode of tachyarrhythmia and sinus tachycardia begin on the 1st day of AMI.16

MATERIALS AND METHODS

This study was a cross-sectional one with 2147 patients...
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A total of 2147 patients aged 35-70 years suffering from acute myocardial infarction (AMI), hospitalized in coronary care units in 1993-1994 were enrolled and followed for cardiovascular death during their hospitalization period. Patients suffering from complicated AMI such as those with arrhythmia, heart failure, cardiogenic shock, pulmonary edema, pericarditis and persistent chest pain were excluded according to physical examination and chest x-ray. Each completed a questionnaire for cardiovascular disease risk factors and, to determine the heart rate, an electrocardiogram was recorded at paper speed of 2.5 cm/s on the 2nd, 5th and final days of hospitalization.

All patients were followed for cardiovascular deaths during their hospitalization period. Cardiovascular death events include sudden coronary death, stroke death and death from arrhythmia or cardiac failure.

All data were coded, checked and entered using the software package Epi 6 data sets at the Computer Unit of the Cardiovascular Research Center in Isfahan. Calculations were done on a personal computer using the statistical software package SPSS. The effect of some variables such as age, sex, infarction type or drugs used were adjusted. All patients were distributed into four groups according to their heart rate (mean of three readings) and mortality rate was determined. The mortality rate was computed for each approximate quartile.

<table>
<thead>
<tr>
<th>Heart rate group * (bpm)</th>
<th>No. of patients</th>
<th>No. expired</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>41-60</td>
<td>132</td>
<td>2</td>
<td>1.5</td>
</tr>
<tr>
<td>61-80</td>
<td>960</td>
<td>31</td>
<td>3.2</td>
</tr>
<tr>
<td>81-100</td>
<td>982</td>
<td>45</td>
<td>4.6</td>
</tr>
<tr>
<td>101-120</td>
<td>73</td>
<td>8</td>
<td>10.9</td>
</tr>
</tbody>
</table>

* Mean of three measurements on the 2nd, 5th and final day of hospitalization.

Fig. 1. Regression relationship between heart rate and mortality rate of 2147 CCU patients.

Cardiovascular disease. In contrast to these positive findings, three large studies found no significant association between heart rate and incidence of CHD. Some reported that cardiovascular-related mortality rate is higher in men than in women irrespective of heart rate, while others consider sinus tachycardia to be a predictor of overall mortality in women only.

The heart rate in patients with acute myocardial infarction (AMI) may vary from marked bradycardia to rapid regular or irregular tachycardia, depending on the underlying rhythm and the degree of left ventricular failure. In this study, as mentioned before, although patients with heart failure, pericarditis, arrhythmia, and persistent chest pain were excluded, mortality still increased as heart rates increased. Therefore it seems that sinus tachycardia can be an independent predictor of mortality in patients with AMI.

Initially after AMI the pulse is rapid and regular (sinus tachycardia at 100 to 110 beats/min), but as the patient's pain and anxiety are relieved, the pulse rate tends to decrease. In order to relieve this initial effect, we measured the heart rate on the 2nd, 5th and last day before discharge, and mean values were categorized in one of 4 quartiles.

The presence of an increased heart rate following AMI is common and considered as an unfavorable prognostic sign because increased heart rate enhances myocardial oxygen demand, while the decrease in diastole decreases...
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diastolic coronary flow. There was no significant difference in the frequency of atrial tachyarrhythmia in relation to the area of AMI. The obtained results from this study were similar, because the type of infarction was adjusted initially.

Several etiologies for atrial tachyarrhythmias after AMI have been proposed, including congestive heart failure and cardiogenic shock, infarct size, compromise of the sinoatrial node blood supply and pericarditis. These causes were excluded in our study and non-complicated patients were enrolled. Another explanation for sinus tachycardia after AMI which was not considered in our study was acute systolic hypertension (ASH). Some studies have reported an increased incidence of acute left ventricular failure and of arrhythmias such as sinus and ventricular tachycardia in patients with ASH in comparison with those with normal blood pressures.

In the coronary care unit, some forms of supraventricular tachyarrhythmias (i.e., sinus tachycardia) are exhibited by nearly two-thirds of patients. On hospital discharge 2 or 3 weeks after the acute event, 30 to 50% of patients still show some type of supraventricular tachyarrhythmia. Supraventricular tachycardia usually develops in the presence of severe hemodynamic decompensation and is associated with an increased risk of subsequent cardiac death. The nonlinear regression relationship obtained in our study indicates that when heart rate was between 101-120 bpm, the mortality rate increases to 10.9%.

Some reports have shown that sinus tachycardia and some types of arrhythmias are the most common clinical and electrocardiographic prodromes of ventricular fibrillation. Unfortunately, this was not investigated in our study.

Sinus tachycardia has been considered as a risk factor for mortality after using thrombolytic therapy in patients with AMI, and for ventricular tachycardia with higher rates (181 to 220 bpm). Some investigators consider sinus tachycardia as a complication of AMI, just as ventricular tachycardia or fibrillation, pulmonary edema, cardiogenic shock and advanced heart block are others. Previous findings from longitudinal studies suggested that persistent sinus tachycardia occurring during the first four hospital days in patients with AMI can predict subsequent complications requiring urgent medical attention, but we could find no similar effect due to the type of our study which was cross-sectional and without patient follow-up.

Sinus tachycardia is considered as one of the later arrhythmias (occurring after the 15th day of AMI) which is controlled by beta-adrenergic blocking agents as well as amiodarone and verapamil. One explanation for the association between heart rate and mortality is the effect of major risk factors. Heart rate was positively correlated with systolic and diastolic blood pressure and cigarette use, and showed a small or inconsistent association with age, serum cholesterol and relative weight. Unfortunately his subject was not investigated in our study.

We conclude that although a strong positive relationship between heart rate and mortality after AMI was obtained from this study, further longitudinal studies with patient follow-up are required in order to improve the predictive ability of sinus tachycardia as an independent predictor of mortality after AMI.

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REFERENCES

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